Dental Defects of Congenital Syphilis

SIMON HILLSON,* CAROLINE GRIGSON, AND SANDRA BOND
Institute of Archaeology, University College London, London, UK

KEY WORDS enamel hypoplasia; Hutchinson's incisors; Moon's molars; mulberry molars; dental development

ABSTRACT Diagnosis of the congenital form of syphilis is an important part of the palaeopathology of this disease. In theory, there are clear clinical signs to be found in the long bones and teeth, but it has rarely been possible to recognise the latter with a confidence in archaeological material, partly because the original descriptions of the dental deformities are sometimes contradictory and partly because it is nowadays difficult to find reference specimens in museums. This article describes two such specimens which have recently been rediscovered, and discusses the form of the dental defects which they show (Hutchinson's incisors, Moon's molars, and mulberry molars) in relation to the developmental sequence of the teeth. Am J Phys Anthropol 107:25–40, 1998. © 1998 Wiley-Liss, Inc.

The purpose of this article is to discuss the value of the classic dental stigmata in palaeopathological diagnosis of congenital syphilis, to examine the mechanisms through which they are generated, and to debate the implications that these have for understanding the sequence of tooth crown development in children. Detailed accounts of these particular tooth crown anomalies are rare, and the article is intended to act as a point of reference for other workers, by describing and illustrating two documented museum specimens from the Royal College of Surgeons of England. Congenital syphilis is the infection of the unborn foetus across the placenta by *Treponema pallidum* from a syphilitic mother. It is considered a rare disease by most clinicians in Europe or America today, due to the maintenance of control through treatment following routine testing of expectant mothers, although it is still common in developing countries (Davanzo et al., 1992) and there have been recent increases among some social groups in large American cities (Berry and Dajani, 1992; Jonna et al., 1995; Rawstron et al., 1993). During the 17th Century AD, however, syphilis was a major scourge (Quetel, 1990) and caused a level of concern comparable to the modern AIDS epidemic, with which it has many parallels (Evans and Frenkel, 1994).

CLINICAL SIGNS OF CONGENITAL SYPHILIS

When a mother has syphilis, the effect on the unborn child is in fact rather variable. Both the mother and child may show no physical symptoms of syphilis at birth, and a proportion of cases register negative in serological testing (Chhabra et al., 1993). Rawstron and colleagues (1993) studied the records of 403 pregnancies, of which they considered 233 to be "at risk" of resulting in an infant with congenital syphilis because the mothers' records showed no history of therapy, but nonetheless noted positive serological findings suggestive of recently acquired syphilis. Out of the 233 at risk, 75 babies were delivered with congenital syphilis diagnosed on the basis of serological tests. Of the 75 babies testing positive for syphilis, 53% were stillborn, and only 6% "had abnormal physical findings at birth"

*Correspondence to: Dr Simon Hillson, Institute of Archaeology, University College London, 31–34 Gordon Square, London WC1H 0PY, United Kingdom. E-mail: simon.hillson@ucl.ac.uk

Received 10 September 1997; accepted 30 May 1998.

(Rawstron et al., 1993: 727). Most of the clinical signs of congenital syphilis develop later (Curtis and Philpott, 1964; Jeans and Cooke, 1930; Stokes, 1945):

1. Early signs. Between about 1 month after birth and 1 or 2 years, a range of lesions may be seen in the skin and the mucous membranes of the mouth and nose. In addition, the bone and cartilage around the growing zones at the ends of long bones may become inflamed.
2. Late signs. From 2 years after birth, or even more than 30 years later, a further range of lesions may develop. These include keratitis of the cornea (with consequent blindness), a degeneration of the nerves (sometimes leading to deafness), and an inflammation of the synovial membranes of joints.
3. Stigmata. A child with congenital syphilis may be marked for life by deformities of the skull, including "saddle-nose" and bossing of the frontal bone. Inflammation of the tibia results in a characteristic bowing known as "sabre shin." There may also be permanent radial scarring (rhagades) at the corners of the mouth, remaining from the skin inflammation of early congenital syphilis. Finally, there are characteristic defects of the teeth—Hutchinson's incisors, Moon's molars, and mulberry molars (see below). These also, in fact, represent the effects of the initial lesions of early congenital syphilis, even though they only become apparent with the eruption of the permanent incisors and first molars around 6 years of age.

Along with the bone deformities, the dental stigmata are the main way in which congenital syphilis can be diagnosed in archaeological human remains (Cook, 1994; Henneberg and Henneberg, 1994; Ortner and Putschar, 1981; Rose, 1985), and there has been discussion (Condon et al., 1994; Jacobi et al., 1992) of the exact form that they take. A great deal hinges on the original descriptions, some made more than a century ago, because well-documented museum specimens are now very rare.

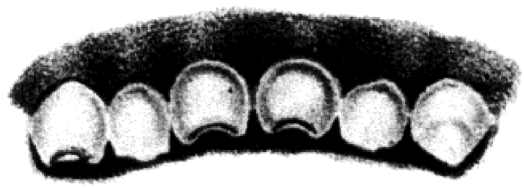


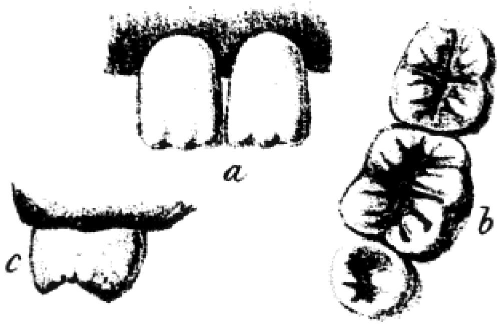
Fig. 1. One of the earliest detailed illustrations of Hutchinson's incisors (Fig. 3 from Plate VI in Hutchinson (1887)). The two permanent upper first incisors have shortened incisal edges, marked with a broad crescentic notch, that appears to have been enlarged by wear which has exposed the underlying dentine. The upper right canine seems to have a deep furrow-shaped defect around its tip "with an excrescence in its centre," whereas the upper second incisors and left canine are unaffected.

THE LITERATURE OF DENTAL STIGMATA IN CONGENITAL SYPHILIS

Sir Jonathan Hutchinson (1857, 1858) was the first to describe defects in the teeth related to congenital syphilis, as part of his triad of signs—dental deformities, keratitis and neural deafness—all of which remain important in the diagnosis of congenital syphilis (Curtis and Philpott, 1964; Jeans and Cooke, 1930). What Hutchinson (1857: 450–451) described was a characteristic deformity of the anterior teeth, particularly permanent incisors (Figs. 1, 2), distinguished by the following features of the crown:

a. Smallness. They were small and rounded in form, and widely set apart.
b. Notching. There was a broad, shallow notch in their incisal edge.
c. Wearing down. They wore away rapidly, removing evidence of the notch.
d. Colour. They had a dirty, greyish enamel surface.

Hutchinson also noted the important point that this particular form of defect was not found on deciduous teeth, although he found that these were often small, of "bad colour," and liable to decay. The great French syphilologist Alfred Fournier (1884) gave a more detailed description of Hutchinson's incisors, noting the crescentic notch in the incisal edge as the most important feature, but also recognising that it was soon lost through wear so that a young adult might show little evidence of it. He also noted (Fournier, 1884:



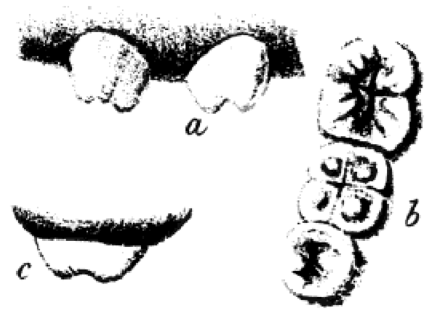


Fig. 2. The original illustration of Moon's molars (Figs. 12 and 13 of Plate IV, in Moon (1877)). Moon's Figure 12 on the left shows a normal permanent lower left first molar in occlusal (12b) and mesial (12c) views, whereas his Figure 13 on the right shows similar views of the defective tooth. Figure 13 is clearly the "bud molar" mesial outline described by Pflüger (1924), but 13b is apparently designed to emphasise the closeness of the cusps in occlusal view and, in doing this, the artist has made them look a little like the cusp nodules of the very different Fournier's molar or Karnosh's "mulberry molar" (see Fig. 3, below). Moon's (1877) accompanying text suggests that he was not intending to illustrate the latter defect, and his Figure 13b may well be the origin of some of the confusion seen in the literature. Also illustrated in Moon's figures are normal incisors (12a) and Hutchinson's incisors (13a).

22) how the length of the incisal edge was drawn in, creating a screwdriver-tip-shaped tooth crown "broader on a level with its neck than at its free edge." Fournier made it clear that the permanent upper first incisors were the main teeth in which these defects were seen. Tantalisingly, he also pointed out that the notch was not necessarily apparent in the teeth when first erupting into the mouth, but gave little detail about this: "At the beginning this notch presents itself, either filled, or in part occupied, with small, apparently atrophied vegetations of the dental tissue..." and "...after a few years they completely disappear, leaving in their place a smooth crescentic notch" (Fournier, 1884: 22–23). It is thus unclear whether or not the central part of the incisal edge was initially covered with enamel, or the underlying dentine lay exposed even before any wear had taken place. Later descriptions did not make it much clearer. Quinlan (1927: 606) stated that "the middle denticle is repressed, and the two lateral denticles proceed in an orderly fashion." Bradlaw (1953: 147) found that "a minute tubercle of enamel may project from the center of the notch...or there may be a thin web of dentine across it," and that "...occasionally it appears as a deep fissure or infolding on both labial and lingual aspects of the enamel." The deformity thus clearly shows some variation between congenital syphilis patients. Putkonen (1962: 47) decided that "the requirement for a syphilitic upper central incisor was barrel shape or convergence of both lateral margins towards the cutting edge," but also observed that:

1. Some teeth bore a distinct crescentic notch at the incisal edge.
2. Other teeth bore, instead of the incisal edge notch, a slight depression on the labial surface, just next to the incisal edge.
3. Still other teeth displayed both the incisal notch and labial depression together.
4. A proportion of teeth had the incisal edge worn flat but, in some of these, the labial depression was still visible. A series of dental impressions from one child showed how the clear notch seen at 7 years of age was much reduced by 9 years, although the labial depression was still visible in a photograph taken at 13 years.
5. Some unworn teeth showed a minute tubercle of enamel in the middle of the incisal edge, with taller lobes rising to mesial and to distal of this, to make the notch. This contrasts with the normal arrangement of three approximately equal sized tubercles, called mamelons, along the incisal edge of incisors.

6. Other unworn teeth showed a crescentic patch of poorly formed enamel at the centre of the incisal edge and, in others, the enamel was missing from this area.

These changes were seen only in the permanent incisors (*not* the deciduous incisors), and only those permanent teeth "in which calcification starts during the first year of life" (Putkonen, 1962: 47). This is the reason that permanent upper lateral incisors (also known as upper second incisors) are not involved, because their crowns start to form much later than the other permanent incisors, towards the end of the first year after birth. "It is curious, for example, to meet alongside of notched central incisors the lateral incisors free from all notching" (Fournier, 1884: 24). Both central and lateral permanent lower incisors, however, start to form at about the time of the upper centrals and can show similar defects to those listed above (see the photograph in Southby, 1981), but these are seen "more seldom in the lower incisors" (Putkonen, 1962: 47).

Fournier (1884) carefully considered Hutchinson's incisors against a background of more general enamel hypoplasia in the anterior teeth, which might affect any part of the crown of any tooth. Such defects include bands of tiny pits, a deep step, or merely a furrow or series of furrows, running around the crown side (Hillson, 1992, 1996; Hillson and Bond, 1997). Fournier concluded that Hutchinson's incisor was the only form of defect which was valuable for diagnostic purposes, because all others could be initiated by a variety of growth-disrupting factors, possibly including syphilis, occurring at any time during the period of tooth crown formation (roughly the first 15 years of life, if the third molar is included). Matters were complicated, however by the influential papers of Cavallaro (1908, 1909), who suggested that a whole variety of defects down the sides of permanent incisor crowns was also related to congenital syphilis and, further, that crescentic notches were frequently found in deciduous incisors, as well as permanent. This latter idea was laid to rest by Karnosh (1926), and it is now clear that Hutchinson's incisors are only a feature of the permanent dentition. The question of the relationship between the characteristic dental stigmata of congenital syphilis and the much wider range of hypoplastic defects which occurs quite independently is, however, still being debated (Condon et al., 1994).

Fournier (1884: 20) also described a defect of the permanent canines in which the tip of the crown was marked around by a sharp circular groove leaving it as "a slender conical stump, which appears as if set in the body of the tooth." Canines like this, together with the typical form of the first incisors in the upper dentition (the later-formed upper second incisors are unaffected) are shown in an excellent plate (Fig. 1) from Hutchinson's famous textbook (1887), which was dedicated to Fournier as his friend and fellow syphilologist. Jacobi et al. (1992: 151) described notched canines associated with Hutchinson's incisors, but these might represent the defect described by Fournier above, after the crown tip had worn away.

The first description of an equivalent defect of the permanent first molars, characteristic of congenital syphilis, was given by Henry Moon (1877: 241), who found that they were "exceedingly prone to be smaller and more dome-shaped than usual." He also illustrated this defect (Fig. 2), showing a mesial view in which the cusps are seen to be set closer together than normal, but with no furrow or other defect running around the circumference of the crown. The surface of the tooth crown thus appears smooth in spite of the deformity. These teeth have gone into the literature as "Moon's molars," although they are also sometimes labelled "bud molars" (Jacobi et al., 1992; Putkonen, 1962). The latter term describes quite closely their appearance, and is due to Pflüger (1924: 606): "Während der normale Molar seinen Kleinsten Durchmesser am Zahnhals und seinen grössten im Bereich der Kauhöcker har, ist es bei der Knospenform gerade ungekehrt: die Zahnbasis bildet hier den grössten Durchmesser." [While the normal molar has its smallest diameter at the neck, and its greatest width at the cusps, in the bud-form these are reversed: the base of the tooth has the largest diameter.] A com-

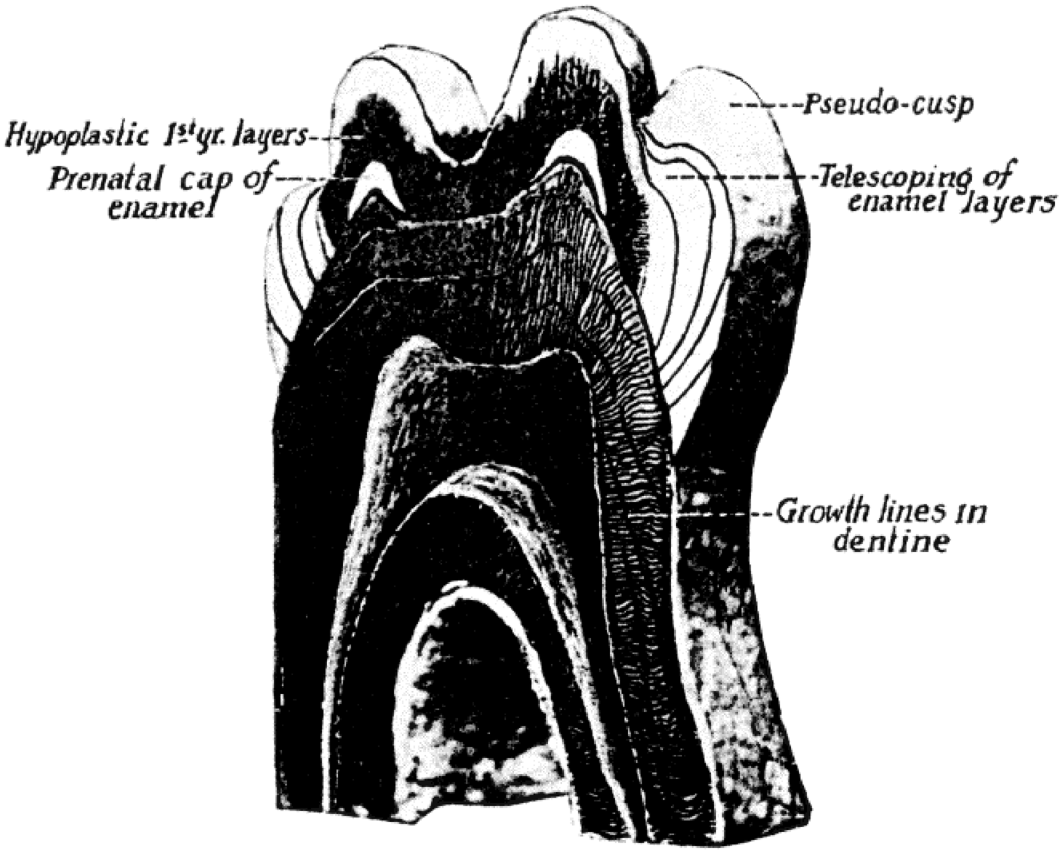


Fig. 3. Reconstruction of a section through a mulberry molar (Fig. 10, p. 39 of Karnosh [1926], reproduced with kind permission from the *Archives of Dermatology & Syphilology,* Volume 13, Copyright 1926, American Medical Association). The plane of section is not clear, but two of the nodules representing the uncompleted cusps are sectioned, surrounded by a deep furrow and a raised rim (marked "pseudo-cusp").

pletely different form of permanent first molar defect, also associated with congenital syphilis, was described by Fournier (1884: 19–20): "The body of the tooth for two-thirds or three-fourths of its height, is in a normal condition; but its upper segment, on the contrary, is lessened in all of its diameters — atrophied, eaten, as it were; separated by a circular furrow, as though it were set in it. At first sight one would say it was a smaller tooth growing out of a larger one, or better still, 'a stump of dentine growing from a normal crown' (Magitot)." This Fournier molar is clearly a prominent defect of enamel hypoplasia, producing a deep furrow around the base of each cusp. Just as clearly, it is a very different defect to that described by Moon and Pflüger, but there is considerable confusion in the literature over these terms. This is largely due to the important paper of Karnosh (1926: 33), in which the deformed permanent first molar of congenital syphilis is "referred to as a mulberry molar of Fournier or Moon." The subsequent description, photographs and diagram (Fig. 3) make it clear that the defect Karnosh was referring to was that of Fournier and not that of Moon or Pflüger: "The whole occlusal surface is rough, pigmented and pinched, with the exception of two or three nodules of fairly good enamel huddled together which represents the poorly developed cusps. All around this occlusal deformity, the enamel of sound formation wells out in the form of a broad collar to the normal size of the crown at the neck of the tooth." (Karnosh, 1926:

34). Once again, this is a marked defect of enamel hypoplasia, running around the base of all cusps. It corresponds to what has been described as a plane-form hypoplastic defect (Hillson and Bond, 1997), in which the uppermost dome-shaped layers of enamel matrix that normally complete the cusps of the crown are missing, to expose the surface of a layer that would normally be buried deep under the cusp tips, with a pronounced step or groove marking the resumption of normal enamel matrix formation below the defect (Hillson, 1996). Such defects may arise from a variety of growth-disrupting factors, but they are rarely so pronounced as this. Karnosh's photographs and diagrams do not illustrate any additional prominent hypoplastic defects down the crown sides, so his "mulberry molar" appears to represent just one major growth disruption.

In summary, therefore, congenital syphilis appears to be associated with some characteristic, and distinct, deformities or stigmata of the permanent dentition:

1. Hutchinson's incisors—primarily seen in permanent upper first incisors, but sometimes also in some permanent lower first or second incisors (never in permanent upper second incisors or canines). Showing a shortened incisal edge, with the mesial and distal crown sides bulging out below it, and marked by a notch of variable shape.
2. Permanent upper or lower canines with a sharp groove-like hypoplastic defect around the tip of their single cusp, as described by Fournier (above).
3. Moon's molars, or bud molars. Only affecting upper and lower permanent first molars. All cusps are abnormally closely spaced, giving a narrow occlusal area relative to the bulge of the crown sides.
4. Fournier molars, or more commonly mulberry molars. Also only affecting permanent first molars. Showing a marked plane-form hypoplastic defect, cutting sharply into the bases of all the cusps.

Possible deformities of the deciduous dentition are more of a problem. Even Cavallaro (1908, 1909) recognised that defects were considerably less common in these teeth. The only real candidate for a characteristic deformity is the occurrence of a pitted hypoplastic defect (Hillson, 1996; Hillson and Bond, 1997) around the base of the cusps in deciduous second molars (Karnosh, 1926). This would certainly conform in development timing to the defects seen in the permanent teeth (see below). The case study of de Wilde (1943) is particularly interesting in this context. Deciduous first incisors from a 3-year-old child with congenital syphilis showed no defect of the enamel, but displayed a prominent ring-like narrowing of the root just below the neck of the tooth. In this position, the disturbance causing the defect would have occurred just after birth (the deciduous first incisor crown is almost complete at birth and the root starts to grow a few weeks afterwards), and microscope examination of tooth sections confirmed that a corresponding zone of defective dentine, inside the root, followed the neonatal line which marked the point of birth (Dean et al., 1993; Hillson, 1996; Schour, 1936).

This question of timing was further addressed by Putkonen (1963), who related the occurrence of Hutchinson's incisors in children with congenital syphilis to the age at which antibiotic treatment was started. Out of 36 patients, only seven displayed clear Hutchinson's incisors, "and these all belonged to the group of 15 whose treatment was not started before they were 3 months old" (Putkonen, 1963: 245). This implies that the disruptions to dental development associated with congenital syphilis most commonly occurred during the first few months after birth, a suggestion that matches the likely timing of the defects in relation to the sequence of tooth crown formation (see below).

DESCRIPTION OF SPECIMENS

At one time, specimens of Hutchinson's incisors, Moon's molars, and mulberry molars appeared routinely in practical classes for dental students but as congenital syphilis became rare in general practice, they were put to one side or thrown away. As a result, it is nowadays difficult to find well-documented specimens, and few anthropologists or palaeopathologists have seen them. Two specimens are, however, available in the Odontological Museum of the Royal College of Surgeons of England, and the purpose of the present article is to describe and

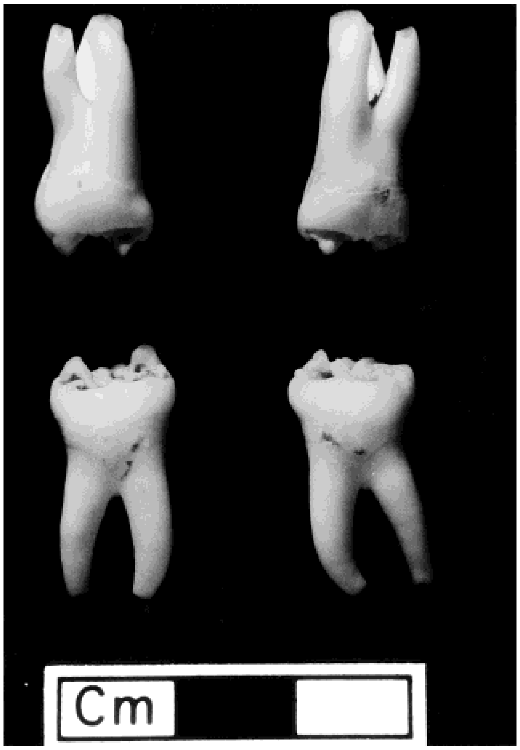


Fig. 4. Permanent first molars showing the Fournier, or mulberry molar defect, from Odontological Museum specimen D33.633, seen in buccal view. The two upper molars are on the top row (the upper right tooth is on the left of the picture), and the lower molars are on the bottom row (the lower right molar is on the left of the picture also). Figures 4–9 are reproduced by kind permission of the Royal College of Surgeons of England.

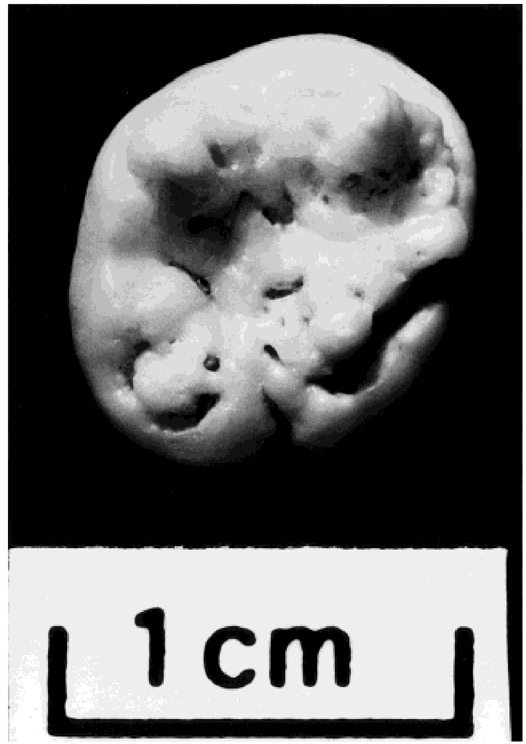


Fig. 5. Occlusal view of the permanent upper right first molar from specimen D33.633. The buccal side is at the top and the mesial side to the right.

illustrate them as a point of reference for other workers. Detailed examination of the surface was possible, but the rarity of these specimens meant that they could not be sectioned.

Catalogue number D33.633, mulberry molars

Description. This specimen comprises the upper and lower left and right permanent first molars (Fig. 4) of one individual, "from a male aged 9 years the subject of congenital syphilis" (catalogue entry). The roots are still not quite completed, as would be expected of first molars extracted at the stated age. In the upper molars, all four cusps are marked by a sharp groove, defining the edge of the hypoplastic defect. The upper right molar is illustrated in detail (Fig. 5) and in this tooth the mesiobuccal (Fig. 6B) and distobuccal (Fig. 6A) cusps are represented by large nodule-like structures, with a sharp, undercut groove at their base on the buccal side, running round to the mesial side of the mesiobuccal cusp, and distal side of the distobuccal cusp. The lower rim of this groove bulges upwards and then curves over gradually into the crown side. The mesiolingual cusp (Fig. 6D) is outlined by groove defects on its mesial and lingual sides, but its enamel tip has broken away to expose a conical protrusion of the underlying dentine. There is still some enamel remaining at the base of the nodule and this is connected to the mesiobuccal cusp by a flange-like structure, also undercut by the groove defect which represents the developing mesial marginal ridge. All three large cusps of the upper molar trigon—the mesiobuccal, mesiolingual, and distobuccal—are therefore united by the occlusal area in between them, and the groove defect marks mainly the distobuccal corner, buccal side, mesiobuccal corner, mesial side, and mesiolingual corner

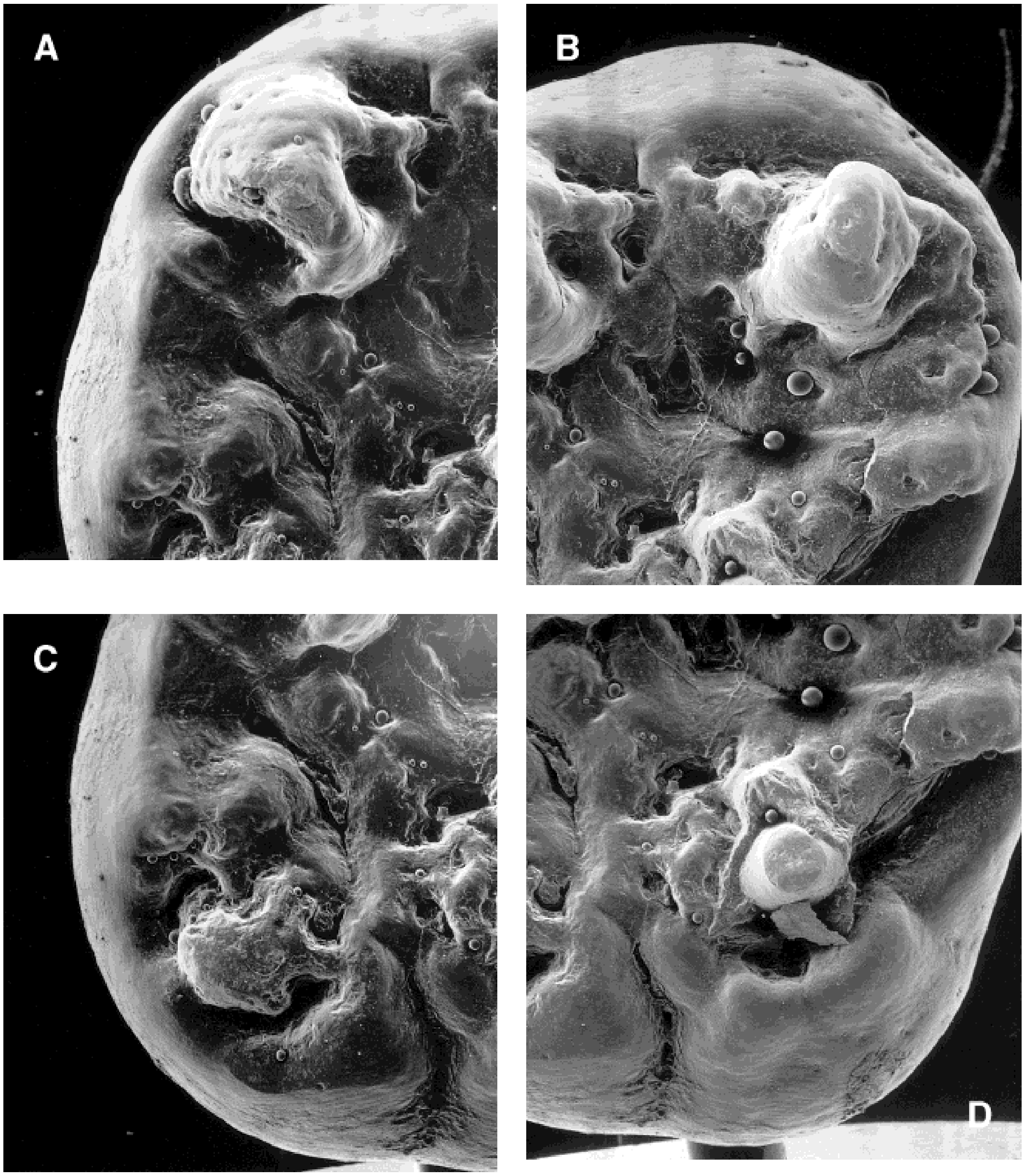


Fig. 6. More detailed occlusal views of the four individual cusps in the specimen shown in Figure 5. (**A**) Distobuccal cusp. (**B**) Mesiobuccal cusp. (**C**) Distolingual cusp. (**D**) Mesiolingual cusp. Epoxy resin replica, examined in a Hitachi S570 scanning electron microscope, using an Everhart-Thornley detector configuration, operated at 20 kV. Field width for each image is 5.8 mm. Note the bubbles, which are difficult to avoid with this technique.

of this trigon. The distolingual cusp (Fig. 6C) nodule is much smaller than the other three, and is almost completely surrounded by the groove defect, although it is united to the occlusal surface by narrow, bridge-like structures. The main furrow defect of the trigon of larger cusps is similarly interrupted by bridges of more normal enamel, most notably at the distobuccal corner (Fig. 7A), but also at the middle of the buccal and mesial sides.

Under the scanning electron microscope, the surface of the crown side below the defect is marked in the normal way by perikymata (Fig. 7), which are the external expression of the regular layering of enamel

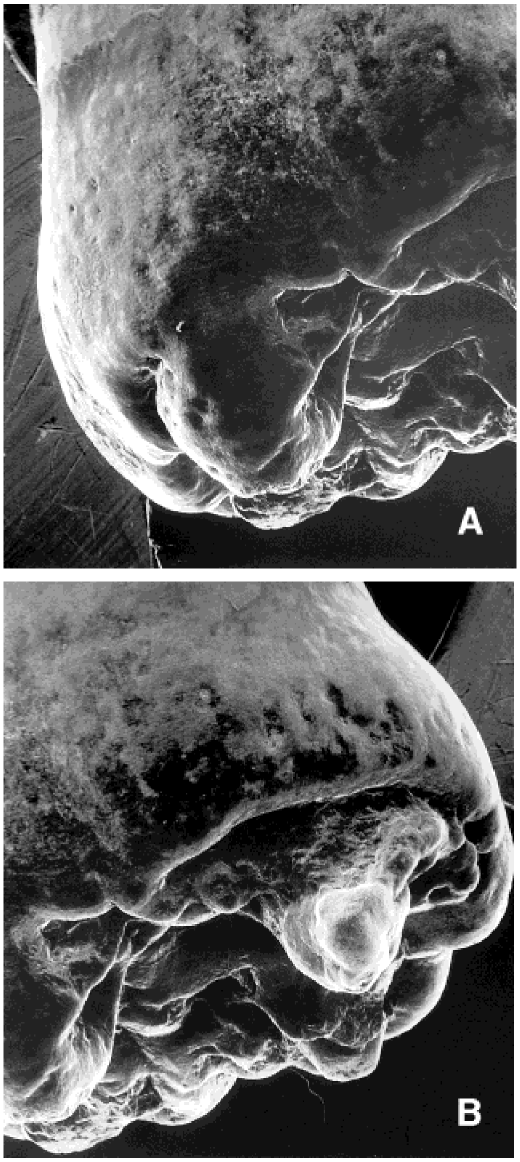


Fig. 7. Buccal views of the specimen shown in Figure 5, with the occlusal surface facing downwards. (**A**) Buccal crown side and distobuccal cusp, with a deep furrow around its base interrupted by a "bridge" of more normal enamel, marked by the fine lines of the perikymata. (**B**) Mesiobuccal cusp with deep furrow around its base, and regular perikymata on the crown side below. Original, uncoated specimen, examined in a Hitachi S570 scanning electron microscope using the Everhart-Thornley detector, operated at 2 kV with a large tilt angle to minimise charging problems (Goldstein et al., 1992). Field width for each image is 5.8 mm.

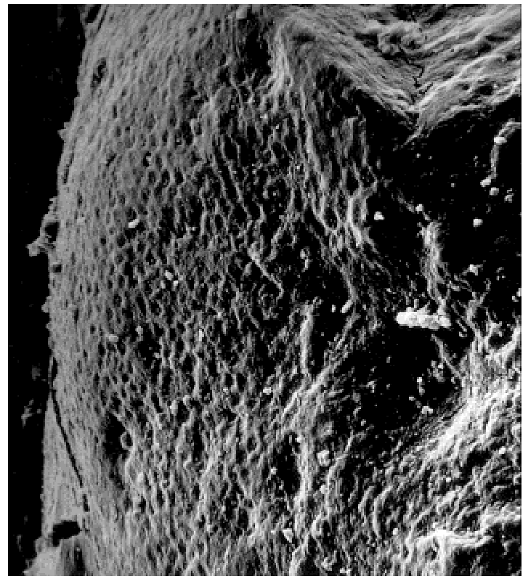


Fig. 8. Detail of the distal surface of the nodule representing the distolingual cusp of the specimen shown in Figure 5. The whole surface is dotted with small circular depressions (each about 4 µm in diameter), which are the Tomes' process pits marking the positions of enamel matrix-forming cells at the point when the disruption to crown development occurred. Epoxy resin replica, examined in a Hitachi S570 scanning electron microscope, using the Everhart-Thornley detector configuration, operated at 20 kV. Field width 220 µm.

formation in the crown (Hillson, 1992, 1996; Hillson and Bond, 1997). They show no evidence of further disruption following the defect at the base of the cusps. The rim of the defect, as it curves over onto the crown side, is also marked by perikymata, as are the bridge-like structures which cross the groove of the defect (Fig. 7A). By contrast, the surface of the cusp nodules does not show the expected perikymata, but is marked all over by Tomes' process pits (Fig. 8), each marking the position of an enamel matrix secreting cell at the point when it was interrupted. In effect, the bulging surface of these cusp nodules marks the layer of enamel matrix which was forming at the time that the defect was initiated.

The lower right molar is similarly illustrated (Fig. 9). In this tooth, the only cusp nodules to survive intact (i.e., they have not broken away) are the mesiolingual and distolingual. They also have their surfaces marked by Tomes' process pits, and have a sharp, undercut groove at their base along the lingual side. On the mesial and distal sides, respectively, there is a bridge-like structure of normal enamel passing down from the cusp nodule, over the defect and onto the crown side. Both of

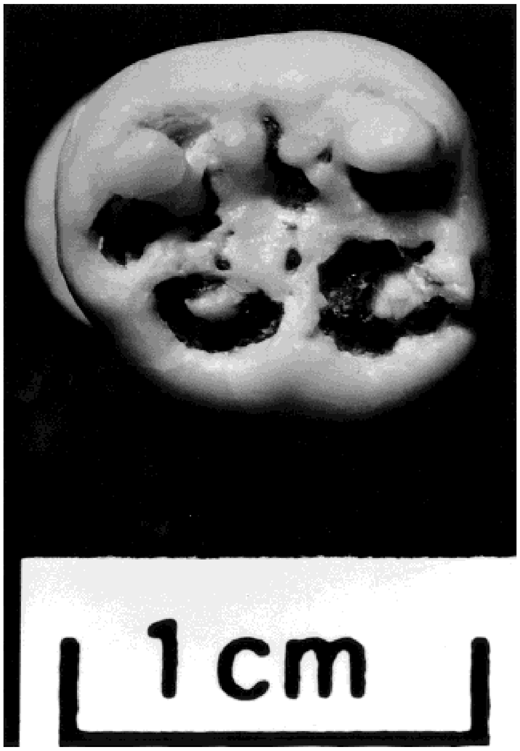


Fig. 9. Occlusal view of the lower right first molar from specimen D33.633. The lingual side is at the top and the mesial side on the right.

these, and the rim marking the lower edge of the defect, show perikymata under the scanning electron microscope. The enamel of the mesiobuccal cusp has fractured away, exposing the underlying dentine, but enough enamel survives at the base to show that it was connected to a flange-like structure at the mesial end of the crown, marking the developing mesial marginal ridge. Cusp nodules are not present for the centrobuccal and distobuccal cusps, although it is not clear whether or not enamel was originally present in these locations, or whether the nodules have fractured away like the mesiobuccal nodule. They are marked by depressions, surrounded by the rim of the groove defect.

The crown diameters for all four molars in this individual, as defined by the main bulge of the side, are within the normal range for permanent first molars. In addition, the spacing of the cusps is normal, in spite of the prominent defect.

Interpretation. This specimen shows the classic mulberry molar or Fournier molar defect described in the literature cited above. In the upper first molar, formation of the tooth crowns was interrupted sharply, before the final enamel matrix increments over the four cusps had been laid down, but after the three cusps of the main trigon had been united to form the outline of the occlusal surface. Here and there, matrix formation continued without interruption, to create the bridge-like structures of normal enamel across the defect, which have been described previously (Boyde, 1970). Enamel matrix formation did not resume over the cusps after the disruption, but it did restart at the base of the cusps, to build first a bulging rim which deepened the defect into a sharp groove, followed by a normal crown side. The early formation sequence of both upper and lower permanent first molars is quite well known from work with tooth germs dissected from aborted foetuses (Butler, 1967a,b; Christensen and Kraus, 1965; Kraus and Jordan, 1965). Dentine and enamel formation are initiated at separate centres underlying the cusps, in a clear order. For upper first molars, the mesiobuccal cusp starts first, followed by the mesiolingual, which is followed rapidly by the distobuccal cusp (although the difference between the last two may be very small), and finally the distolingual cusp. For lower first molars, the order is mesiobuccal first, then mesiolingual, centrobuccal (Hillson, 1996), distolingual, and lastly distobuccal. In both upper and lower permanent first molars, the earliest dentine and enamel matrix formation may start in some foetuses a little before 28 weeks after conception. By 36 weeks after conception, the time normally accepted as "term" (although birth may vary between 34 and 42 weeks after conception), all foetuses show at least dentine and enamel formation in the mesiobuccal cusp, and most show it in the mesiolingual cusp as well, while in a few the distobuccal cusp may have started, too (Christensen and Kraus, 1965). The distolingual cusp has not usually started at this stage. Within one individual, lower first molar development is slightly in advance of upper first molar development. From these timings, it is clear that the defect shown in the mulberry molars of specimen D33.633

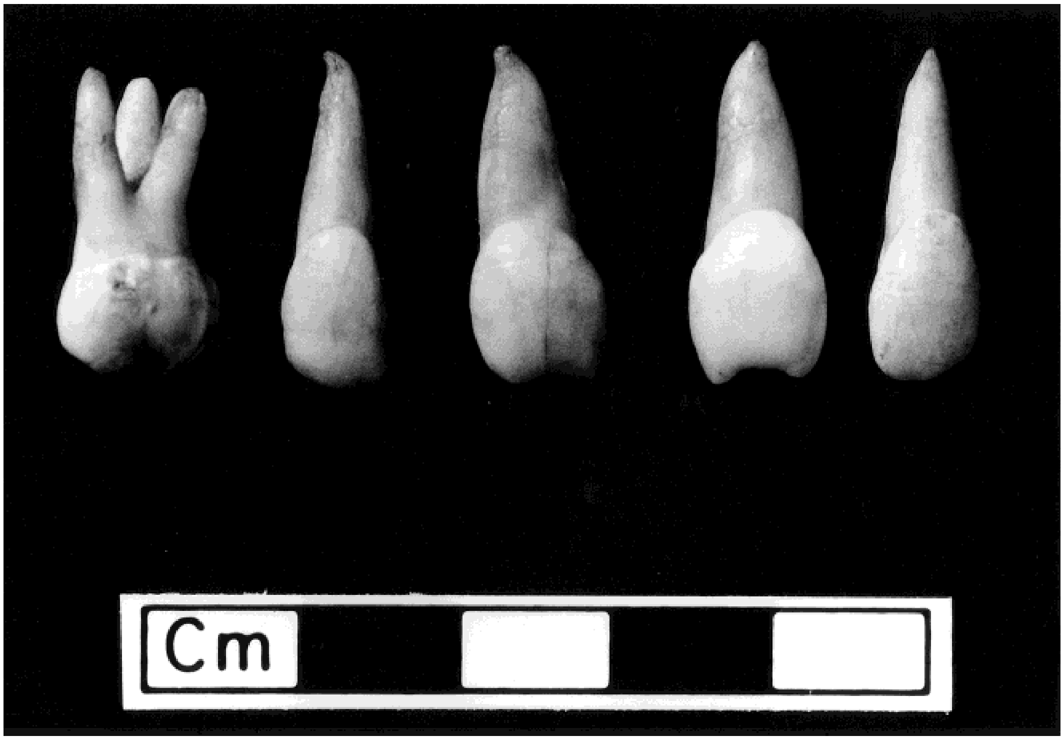


Fig. 10. Odontological Museum specimen D33.632; labial/buccal views of Moon's molar and Hutchinson's incisors. Arranged from left to right in the following order—permanent upper left first molar, upper right second (lateral) incisor and first (central) incisor, upper left first incisor and second incisor. Figures 10 to 14 are reproduced by kind permission of the Royal College of Surgeons of England.

described here is extremely unlikely to have been caused before birth and, in fact, probably occurred some weeks after birth, although the interval cannot be estimated simply. It is assumed that the defect is related to the subject's congenital syphilis condition, although there is no direct proof of this because there is no evidence of any major growth disruption either before or after the main defect.

Catalogue number D33.632, Hutchinson's incisors and Moon's molar

Description. This specimen comprises the upper right and left permanent first and second incisors, with the upper left permanent first molar, from one individual (Fig. 10). It was presented by Sir Frank Colyer, who established most of the Odontological Museum collections during the first part of the present century, and is best known for his *Variations and Diseases of the Teeth of Animals* (Colyer, 1936; Miles and Grigson, 1990).

The incisal edge of the upper first incisors (Fig. 11) is shortened, and the mesial and distal crown sides bulge out below it, giving the labial outline of the crown the pumpkin seed-shaped quality described by Jacobi and colleagues (1992). Considerable occlusal wear has created a curved facet at the incisal edge, exposing a broad band of dentine and creating a shallow notch. This wear, however, makes it very difficult to see what the original defect was like. As examined in the scanning electron microscope, the crown side is marked by perikymata which follow a normal contour (Fig. 12). There is no sign of any major defect, although some very minor furrow-form hypoplastic defects can be seen in the cervical third of the crown side. The second incisors (Fig. 10) show no sign of the incisal edge shortening, or notch, although the variation in prominence of the periky-

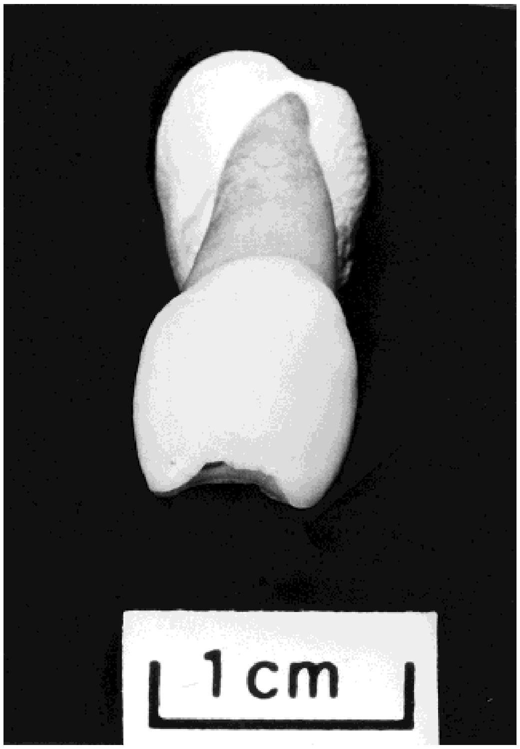


Fig. 11. Upper left incisor from specimen D33.632, showing the labial surface with the incisal edge tilted towards the camera. It shows the characteristic narrowing of the incisor edge, together with a broad notch exposing the dentine, described in the original accounts of Hutchinson's incisor.

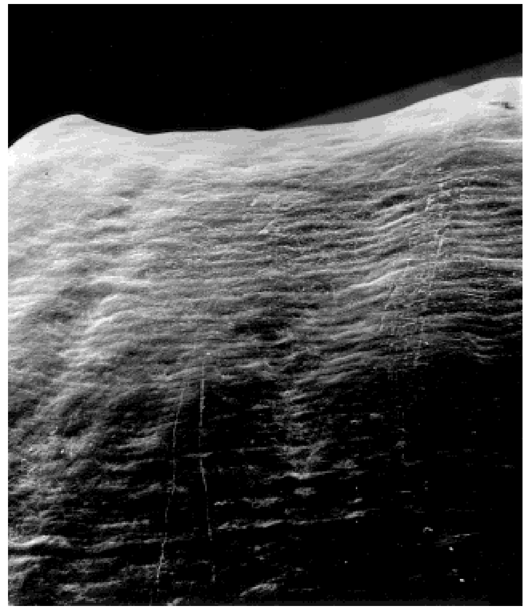


Fig. 12. Labial crown side of the specimen shown in Figure 11, with the incisal edge to the top. The horizontal lines running across the surface are the perikymata which mark normal enamel matrix formation. Some of these are exaggerated into minor furrow-like defects representing the minimum expression of defect seen in the much more common generalised forms of enamel hypoplasia (see text). Epoxy resin replica, examined in a Hitachi S570 scanning electron microscope, using an Everhart-Thornley detector configuration, operated at 20 kV. Field width 4 mm.

mata down the first incisor side can be matched between the parts of the crown sides formed at the same time in these two teeth (Dean et al., 1993; Hillson, 1992, 1996).

The first molar (Figs. 10, 13) shows no sign of any hypoplastic defect, and the crown sides are smooth, with perikymata of normal spacing and contour (Fig. 14). Viewed from the occlusal aspect, however, the cusps are markedly closer together than normal (Fig. 13). The occlusal surface is therefore relatively small, while the maximum diameter of the crown, as defined by the bulge of the crown sides, is normal for an upper first molar. This gives the crown a somewhat spherical form, looking quite like the swollen bud of a plant about to burst.

Interpretation. The upper first incisors correspond closely to the descriptions of Hutchinson (1858) and Fournier (1884) for the deformity now commonly known as Hutchinson's incisor. The pattern of wear has presumably exploited the original defect to produce a distinctive notch, and the drawing-in of the mesial and distal crown sides adds to the distinctive form. It would only take a little more wear, however, to remove enough of these features to render diagnosis impossible. No details of the original form of the incisal edge are preserved, although enough of the crown side remains to show that the growth disruption responsible for the deformity occurred in the very first part of the development sequence of the tooth. Little is known of the incisor development sequence, but work with the tooth germs of deciduous incisors from aborted foetuses (Kraus, 1959) has shown that the central part of the incisal edge is the first to start formation of dentine and enamel matrix. Normal incisors have three main lobes called

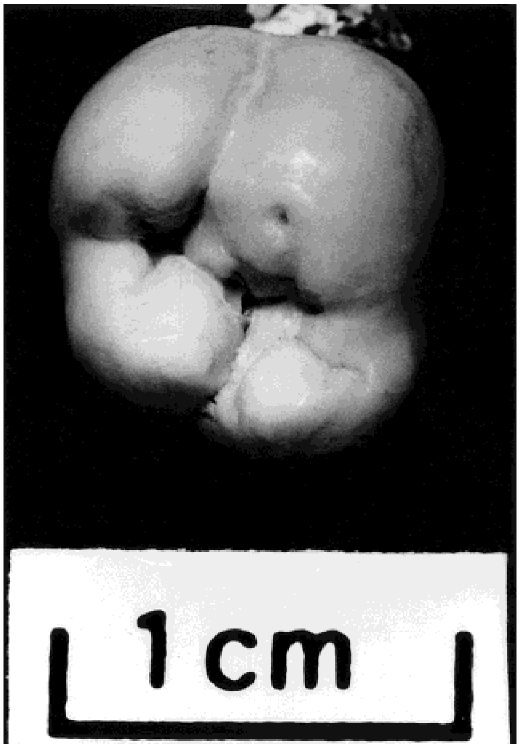


Fig. 13. Occlusal view of upper left first molar from specimen D33.632, showing the crowding together of the four cusps. The buccal side is at the top and the mesial side to the left.

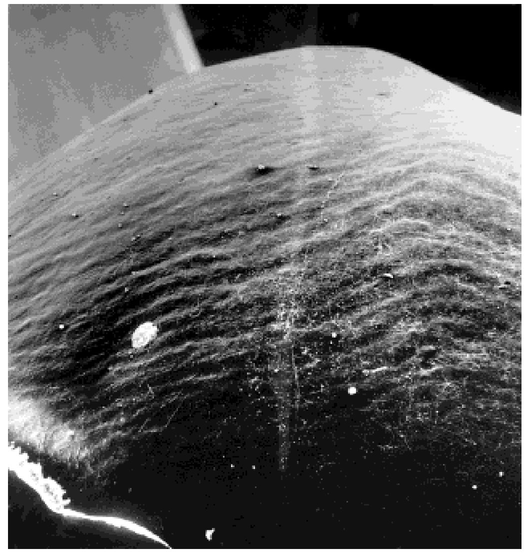


Fig. 14. Buccal side of the distobuccal cusp in the specimen shown in Figure 13, with the cusp tip at the top. The horizontal lines are the perikymata which mark normal enamel matrix formation from the tips of the cusps down to the cervical part of this tooth. Epoxy resin replica, examined in a Hitachi S570 scanning electron microscope, using the Everhart-Thornley detector, operated at 20 kV. Field width 250 µm.

mamelons on the incisal edge (rather rapidly worn away but visible in newly erupted teeth), and the first mineralised tissue is laid down under the central one of these. As this development centre grows, it forms "shoulders" that ultimately develop into the mesial and distal mamelons, which thus do not develop from separate centres. The logical explanation of the notch in Hutchinson's incisors is that a disruption to the earliest formation of the crown, under the central mamelon, caused the later formed mesial and distal mamelons to become more closely spaced than normal. The timing of this, however, is unclear. Most studies of the postnatal development of teeth are based upon radiographs, and various constraints on this technique have meant that the early development of the incisors is poorly covered (Anderson et al., 1976; Fanning and Brown, 1971; Haavikko, 1970; Moorrees et al., 1963). The widely used table of Schour and Massler (1940) suggests that upper first incisors, and lower first and second incisors, start to form between 3 and 4 months after birth, but it is unclear what these figures are based upon (Smith, 1991). Recently, a histological study of incremental structures in several teeth from a single individual suggested that the lower second incisor started to form around the time of birth (Dean et al., 1993). This would imply that enamel formation in the upper first incisors might, if anything, start slightly before birth in some individuals, although none of the literature describes a neonatal line (marking the point of birth) in permanent incisors—such lines are consistently described in permanent first molars and all deciduous teeth (Bouyssou et al., 1957; Dean et al., 1993; Schour, 1936). In any case, for the specimen described here it is not possible to see whether or not any enamel matrix had been secreted before the growth disruption occurred, because this part has been worn away and no clear conclusions can be drawn.

The fact that no hypoplastic defect is visible on the surface of the cuspal enamel in

the first molar suggests that no serious disruption occurred during the formation of the underlying enamel matrix. It would be expected that any such disruption would show itself by either a plane-form or a pit-form defect in this part of the crown. This in turn implies that whatever caused the cusps to be spaced abnormally close together occurred before the onset of enamel matrix formation in this tooth. Unfortunately, this is the only Moon's molar in the collection of the Royal College of Surgeons, and it is not possible to section it. The exact nature of the defect which causes the deformity of Moon's molars cannot, therefore, be determined until further material becomes available. It could either be a disruption to the morphogenesis of the tooth germ before deposition of dentine and enamel matrix was started, or some variation in the thickness of the buried increments of enamel matrix, as suggested above. The latter, however, might be expected to produce some deficiency on the outer surfaces of the cusps, as well as on the sides facing the occlusal surface, and this has not happened.

DISCUSSION

The first question to be addressed is whether or not these deformities of the teeth can be used to diagnose congenital syphilis in palaeopathology, or whether they overlap the wide range of defects of enamel hypoplasia seen in individuals with no history of the disease (Condon et al., 1994). The specimens of mulberry molars described here clearly overlap with other plane-form hypoplastic defects, which could be caused by a whole range of growth-disturbing factors, so this deformity cannot be seen as pathognomonic of congenital syphilis. Putkonen (1962) excluded such teeth from a clinical study of congenital syphilis patients in Finland. The detailed form of the defect, however, is at least unusual in its location and the sharpness of the furrow marking its edge, implying a particularly abrupt resumption of enamel matrix formation. This in itself can be seen in other defects from time to time but, combined with the location of the defect at a particularly early stage in the crown formation sequence, it would at least be suggestive of congenital syphilis.

The Hutchinson's incisors and Moon's molars described here are unlike any other defect and, in such a pronounced form, should be pathognomonic of congenital syphilis. Putkonen (1962) found that, out of 235 patients treated for congenital syphilis, 45% of those with permanent first incisors had the clear syphilitic form by his definition (above) and 22% of those with first molars had definite Moon's molars. This contrasted with only 12% of patients whose limbs were radiographed, showing evidence of cortical thickening relating to periostitis. Dental stigmata would, therefore, seem to have a diagnostic advantage over osseous deformities, and the teeth are usually better preserved than bones at archaeological sites. The difficulty may lie in variation, which cannot, of course, be investigated with one specimen, and the fact that even a little occlusal wear would remove many of the diagnostic features. Some studies, for example Putkonen (1962, 1963) or Sarnat and Shaw (1942), have recorded a "possibly syphilitic" category of incisors when the incisal edge has been worn flat and there is no evidence of the notch, but it is still possible to see convergence of the mesial and distal sides. This is difficult, because there is normal variation in the labial outline of the upper first incisors (Jordan et al., 1992), and it would not provide strong evidence for diagnostic purposes.

What has never been clear, however, is the precise nature of the disruption to tooth crown formation which is caused by congenital syphilis, and the reason for its very short duration. Most infants with congenital syphilis show no visible signs of the disease at the time of birth, and some weeks may elapse before they do, although the majority start to show some symptoms by 2 months after birth (Jeans and Cooke, 1930). Similarly, several weeks may elapse before a positive serological reaction develops in the infant (Parker and Collier, 1990). This is because any antibody involved with immune response in the foetus in utero comes from the mother, and it takes time for the baby to develop its own immune response after birth. Bauer (1944) investigated the histopathology of the jaws in a small number of children with congenital syphilis, including stillborn

foetuses and babies dying soon after birth. He showed convincingly that the enamel organ of the developing tooth germ was invaded by *Treponema pallidum* and that levels of these microorganisms decreased with increasing age of the infant. Histological evidence of inflammatory response in the enamel organ was, however, much less apparent in the stillborn foetuses than it was in the infant specimens. From this, it seems more likely that the crown growth disruption caused by congenital syphilis occurs after birth rather than before. This is borne out by the mulberry molar specimen described here, where the defect clearly occurred some weeks further on in the crown development sequence than would normally be expected at birth. It is less clear why a disease that shows gradual changes in its other clinical manifestations should show such an abrupt and apparently short-lived change in the teeth.

If, as has been suggested above, the Moon's molar described here represents a disruption of the development of the tooth germ before enamel matrix formation started, then this does not necessarily mean that the disruption occurred before birth. Two types of variation need to be borne in mind. First is variation in the time at which permanent first molar dentine and enamel formation start, which may be less than 28 weeks after conception, varying up to 32–36 weeks. Second is the variation in gestation period, because the interval between conception and birth may range from less than 34 weeks to over 42 weeks, and babies with congenital syphilis typically have a low gestational age (Rawstron et al., 1993). It is thus perfectly possible that, in a child born prematurely, the first molars could not yet have started to form. Whatever the timing involved, however, the finding of matching defects in the cusps of the first molar and the central part of the first incisor suggests that they are closer together in the start of their crown development than is proposed by standard tables based upon radiographic evidence (above). This is an important point in the establishment of chronologies for dental development, which are used widely for both forensic and archaeological purposes in determining age-at-death.

CONCLUSIONS

This article presents rare museum specimens showing the dental defects numbered among the classical stigmata of congenital syphilis. They appear to represent a single disruption to tooth crown formation a few weeks after birth, but not in utero, or during the later development of the teeth. It is hoped that more specimens may be found, which could be sectioned in order to investigate these defects further.

ACKNOWLEDGMENTS

The authors thank the President and Council of the Royal College of Surgeons of England for permission to examine, photograph, and take dental impressions of Odontological Museum specimens D33.633 and D33.632, and for permission to publish the images. None of this would have been possible without the extraordinary energy and widespread research interests of Sir Frank Colyer (1866–1954), Honorary Curator of the Odontological Museum at the Royal College of Surgeons from 1900 until his death, who preserved these specimens. Many thanks are also due to Dr. R.S. Morton, Honorary Lecturer in the History of Medicine at the University of Sheffield, who used his long experience as a practising venereologist to advise the authors, and who kindly commented on this paper. The project originally started in the principal author's attempts to find reference material following discussions with Dr. K.W. Condon, of Texas Southwestern Medical Center, involving an attempt to study defects in archaeological material. The authors have further benefitted from the helpful comments of the anonymous reviewers appointed by the American Journal of Physical Anthropology.

LITERATURE CITED

Anderson DL, Thompson GW, and Popovitch F (1976) Age of attainment of mineralisation stages of the permanent dentition. J Forensic Sci *21*:191–200.

Bauer WH (1944) Tooth buds and jaws in patients with congenital syphilis. Correlation between distribution of *Treponema pallidum* and tissue reaction. Am J Pathol *20*:297–319.

Berry MC, and Dajani AS (1992) Resurgence of congenital syphilis. Infect Dis Clin North Am *6*:19–29.

Bouyssou M, Bouyssou H, and Teulie S (1957) L'inlfuence de la naissance et de la periode néo-natale sur les structures dentaire à l'état normal et pathologique. Cah d'Odonto-Stomatol *7*:15–53.

Boyde A (1970) The surface of the enamel in human hypoplastic teeth. Arch Oral Biol *15*:897–898.

Bradlaw RV (1953) The dental stigmata of prenatal syphilis. Oral Pathol *6*:147–158.

Butler PM (1967a) Comparison of the development of the second deciduous molar and first permanent molar in man. Arch Oral Biol *12*:1245–1260.

Butler PM (1967b) Relative growth within the human first upper permanent molar during the prenatal period. Arch Oral Biol *12*:983–992.

Cavallaro J (1908) Syphilis in relation to dentition (Parts A and B). Dent Cosmos *50*:1161–1174, 1325–1344.

Cavallaro J (1909) Syphilis in relation to dentition (Parts C and D). Dent Cosmos *51*:16–34, 156–186.

Chhabra RS, Brion LP, Castro M, Freundlich L, and Glaser JH (1993) Comparison of maternal sera, cord blood, and neonatal sera for detecting presumptive congenital syphilis: Relationship with maternal treatment. Pediatrics *91*:88–91.

Christensen GJ, and Kraus BS (1965) Initial calcification of the human permanent first molar. J Dent Res *44*:1338–1342.

Colyer JF (1936) Variations and Diseases of the Teeth of Animals. London: John Bale, Sons & Danielsson.

Condon K, Becker J, Condon C, and Hoffman JR (1994) Dental and skeletal indicators of a congenital treponematosis. Am J Phys Anthropol Suppl *18*:70.

Cook DC (1994) Dental evidence for congenital syphilis (and its absence) before and after the conquest of the New World. In O Dutour, G Palfi, J-P Brun (eds): The Origin of Syphilis in Europe. Before or After 1493? Toulon: Centre Archeologique du Var, Editions Errance, pp. 169–175.

Curtis AC, and Philpott OS (1964) Prenatal syphilis. Med Clin North Am *48*:707–719.

Davanzo R, Antonio C, Lincetto O, and Schierano S (1992) Neonatal and post-neonatal onset of early congenital syphilis: A report from Mozambique. Ann Trop Paediatr *12*:445–450.

de Wilde H (1943) Stigmata of congenital syphilis in the deciduous dentition. Am J Orthodont Oral Surg *29*: 368–376.

Dean MC, Beynon AD, Thackeray JF, and Macho GA (1993) Histological reconstruction of dental development and age at death of a juvenile *Paranthropus robustus* specimen, SK 63, from Swartkrans, South Africa. Am J Phys Anthropol *91*:401–420.

Evans HE, and Frenkel LD (1994) Congenital syphilis. Clin Perinatol *21*:149–162.

Fanning EA, and Brown T (1971) Primary and permanent tooth development. Aust Dent J *16*:41–43.

Fournier A (1884) Syphilitic teeth. Dent Cosmos *26*:12–25, 141–155.

Goldstein JI, Newbury DE, Echlin P, Joy DC, Romig AD, Lyman CE, Fiori C, and Lifshin E (1992) Scanning Electron Microscopy and X-ray Microanalysis. New York: Plenum Press.

Haavikko K (1970) The formation and the alveolar and clinical eruption of the permanent teeth. Proc Finn Dent Soc *66*:101–170.

Henneberg M, and Henneberg R (1994) Treponematosis in an ancient Greek colony of Metaponto, Southern Italy, 580–250 BCE. In O Dutour, G Palfi, J-P Brun (eds): The Origin of Syphilis in Europe. Before or After 1493? Toulon: Centre Archeologique du Var, Editions Errance, pp. 92–98.

Hillson SW (1992) Dental enamel growth, perikymata and hypoplasia in ancient tooth crowns. J R Soc Med *85*:460–466.

Hillson SW (1996) Dental Anthropology. Cambridge: Cambridge University Press.

Hillson SW, and Bond S (1997) Relationship of enamel hypoplasia to the pattern of tooth crown growth: A discussion. Am J Phys Anthropol *104*:89–104.

Hutchinson J (1857) On the influence of hereditary syphilis on the teeth. Trans Odontol Soc Great Britain *2*:95–106.

Hutchinson J (1858) Report on the effects of infantile syphilis in marring the development of teeth. Trans Pathol Soc London *9*:449–456.

Hutchinson J (1887) Syphilis. London: Cassell & Co.

Jacobi KP, Cook DC, Corruccini RS, and Handler JS (1992) Congenital syphilis in the past: Slaves at Newton Plantation, Barbados, West Indies. Am J Phys Anthropol *89*:145–158.

Jeans PC, and Cooke JV (1930) Prepubescent Syphilis. New York: D. Appleton & Co.

Jonna S, Collins M, Abedin M, Young M, Milteer R, and Beeram M (1995) Postneonatal screening for congenital syphilis. J Fam Pract *41*:286–288.

Jordan RE, Abrams L, and Kraus BS (1992) Kraus' Dental Anatomy and Occlusion. St Louis: Mosby Year Book.

Karnosh LJ (1926) Histopathology of syphilitic hypoplasia of the teeth. Arch Dermatol Syphilol *13*:25–42.

Kraus BS (1959) Calcification of the human deciduous teeth. J Am Dent Assoc *59*:1128–1136.

Kraus BS, and Jordan RE (1965) The Human Dentition Before Birth. Philadelphia: Lea & Febiger.

Miles AEW, and Grigson C, eds. (1990) Colyer's Variations and Diseases of the Teeth of Animals. Cambridge: Cambridge University Press.

Moon H (1877) On irregular and defective tooth development. Trans Odontol Soc Great Britain *9*:223–243.

Moorrees CFA, Fanning EA, and Hunt EE (1963) Age variation of formation stages for ten permanent teeth. J Dent Res *42*:1490–1502.

Ortner D, and Putschar W (1981) Identification of Pathological Conditions in Human Skeletal Remains. Washington: Smithsonian Institution Press.

Parker MT, and Collier LH (1990) Topley and Wilson's Principles of Bacteriology, Virology and Immunity. London: Edward Arnold.

Pflüger H (1924) Eine für Lues congenita charakteristiche Formveränderung (Knospenform) an dem ersten Molaren. Münch Med Wochenschr *71*:605–607.

Putkonen T (1962) Dental changes in congenital syphilis. Relationship to other syphilitic stigmata. Acta Derm Venerol *42*:44–62.

Putkonen T (1963) Does early treatment prevent dental changes in congenital syphilis? Acta Derm Venerol *43*:240–249.

Quetel JC (1990) History of Syphilis. Baltimore: Johns Hopkins University Press.

Quinlan RV (1927) The teeth in cases of congenital syphilis. Arch Dermatol Syphilol *16*:603–610.

Rawstron SA, Jenkins S, Blanchard S, Ping-Wu L, and Bromberg K (1993) Maternal and congenital syphilis in Brooklyn, NY. Am J Dis Child *147*:727–731.

Rose JC (1985) Gone to a Better Land: A Biohistory of a Rural Black Cemetery. Fayetteville: Arkansas Archaeological Society.

Sarnat BG, and Shaw NG (1942) Dental development in congenital syphilis. Am J Dis Child *64*:771–788.

Schour I (1936) Neonatal line in enamel and dentin of human deciduous teeth and first permanent molar. J Am Dent Assoc *23*:1946–1955.

Schour I, and Massler M (1940) Studies in tooth development: The growth pattern of human teeth. J Am Dent Assoc *27*:1778–1792, 1918–1931.

Smith BH (1991) Standards of human tooth formation and dental age assessment. In MA Kelley, CS Larsen (eds): Advances in Dental Anthropology. New York: Wiley-Liss, pp. 143–168.

Southby R (1981) Hutchinson teeth. Aust Paediatr J *17*:226.

Stokes JH (1945) Modern Clinical Syphilology. Philadelphia: WB Saunders.